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A DOSIMETRY MODEL FOR HYGROSCOPIC SULFATE AEROSOLS IN SELECTED TEMPERATURE AND RELATIVE HUMIDITY PATTERNS

by

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16. ABSTRACT

Sulfate aerosols (NH_4HSO_4, $(NH_4)_2SO_4$ and H_2SO_4) are of international health effects concern because of their global prevalence and potential irritant or toxic effects on human health. To assess hazards following inhalation exposure, the total dose delivered to the human respiratory tract and its regional distribution must be determined. It is known that such information can be expressed as a function of the mass median aerodynamic diameter (MMAD) of an inhaled aerosol. Sulfate aerosols are hygroscopic and will have changing particle sizes and densities upon absorption of H_2O vapor in the humid environment of the human respiratory tract. We have developed a new mathematical description of inhaled particle behavior suitable for dosimetry of the human nasopharyngeal and oropharyngeal compartments, larynx, and tracheo-bronchial (TB) airways. For non-hygroscopic aerosols, the dosimetry model predicts the sigmoidal shape of TB deposition patterns experimentally measured in human test subjects, the slopes of the quasi-linear segments of these curves, and the positions of their two asymptotes. We have employed data that describe sulfate particle sizes as a function of relative humidity as computer subroutines of the model to calculate the dose dispersion of NH_4HSO_4, $(NH_4)_2SO_4$ and H_2SO_4 aerosols in man. Their deposition patterns are very different from those of non-hygroscopic aerosols with identical inhaled MMAD values. Results show that the hygroscopic characteristics of ambient sulfate aerosols can greatly affect total dose deposited and the relative distribution of particle mass among airways.

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INTRODUCTION

Atmospheric sulfate aerosols (H_2SO_4, $(NH_4)_2SO_4$, and NH_4HSO_4) from industrial emissions are of international concern because of their global prevalence and potential irritant or toxic effects on human health (Husar *et al.*, 1978). To assess hazards following inhalation exposure, the total dose delivered to the human respiratory tract and its regional distribution must be determined. It is known that such information can be expressed as a function of the mass median aerodynamic diameter (MMAD) of an inhaled aerosol (Task Group on Lung Dynamics, 1966). These sulfate aerosols are hygroscopic and will have changing particle sizes and densities upon absorption of water vapor in the humid environment of the human respiratory tract. We have employed experimental and theoretical data that describe particle sizes as a function of temperature (T) and relative humidity (RH) as computer subroutines of an aerosol deposition model to calculate the dose dispersion of H_2SO_4, $(NH_4)_2SO_4$, and NH_4HSO_4 aerosols in man for two different T and RH environments corresponding approximately to nasal and oral breathing. The predicted deposition patterns are very different from those of nonhygroscopic aerosols with identical inhaled MMAD values.

METHODS

Aerosol Growth Data. The hygroscopic growths of H_2SO_4, $(NH_4)_2SO_4$ and NH_4HSO_4 particles to final equilibrium sizes in precisely defined moist environments have been investigated by Tang and Munkelwitz (1977). Experimental data for NH_4HSO_4 aerosols, and theoretical growth curves for all the aerosols are presented in Figure 1. We shall use the growth curves in Figure 1 to define particle parameters (size and density) as a function of assumed respiratory tract T and RH profiles as discussed below.

Ambient Aerosol Sizes. Chemical composition analyses of urban atmospheres have established that $SO_4^=$ and NO_3^- and NH_4^+ ions are mostly extracted from constituent particulate matter with ambient MMADs in the 0.2-0.6 μm range (Patterson and Wagman, 1977). In the nasopharyngeal region, inspired air becomes about 70% conditioned to the atmospheric environment of the deep lung (Hanna and Scherer, 1982). For a tidal volume of 500 ml, the residence time of an inhaled particle in the nasopharyngeal region will be about 0.09 seconds. Calculations for 12 different salts (Ferron, 1977) have shown that particles of aerodynamic diameter, D_{ae}, < 0.2 μm reach equilibrium diameters at 99.5% RH and 37°C conditions within 0.1 seconds; the time is 2 seconds for 1 μm D_{ae} particles. Submicron ambient H_2SO_4, $(NH_4)_2SO_4$, and NH_4HSO_4 aerosols will grow considerably while in transit to the trachea by becoming equilibrated to a 70% conditioned T and RH environment (see Figure 1).

Temperature and Relative Humidity Profiles In Man. A systematic (airway-by-airway) mapping of T and RH profiles in human test subjects has not been reported in the open literature. Available data on T and RH conditions in man vary greatly; Ingelstedt (1956) extensively reviewed the published literature, explained anomolies and discussed differences between reported findings, and concluded that in future investigations: T and RH data must be taken *in situ* (*i.e.*, not inferred from external observations), T and RH values must be measured simultaneously, and anaesthesia should be avoided if possible. To date, few investigations have satisfied those stringent criteria. Following surgical implantation of instrumentation, Ingelstedt (1956) simultaneously measured T and RH values at a **fixed** location (laryngeal cavity) within numerous unanesthesized humans. For nasal breathing at room conditions (23°C, 30%RH), mean temperatures of 32°C (inspiration) and 36°C (expiration) were recorded; mean RH values were 98-99% during both phases of the respiratory cycle. For oral breathing, mean temperatures were 31°C (inspiration) and 36°C (expiration); mean RH values, however, were very **different** during inspiration and expiration, 90% and 99%, respectively. Ferrus *et al.* (1980) have measured respiratory water loss and have similarily concluded that the overall expired tidal volume is not fully water saturated.

Invasive techniques (Déry *et al.*, 1967) were necessary to record T and RH data at **non-fixed** locations in bronchial airways. They measured profiles in the trachea and large airways of humans (of necessity, anaesthesized) with a flexible microthermistor probe. Mean trachea T and RH values were 31°C and 86% during inspiration. At a depth of 10 cm **below** the main carina, mean T and RH values were 33°C and 91%; assuming the influence of anaesthesia to be general (the use of anaesthesia may cause changes in the production and excretion of mucus (Han and Lowe, 1968)) a RH of ~95% may be proposed at this location in accordance with Ingelstedt's (1956) work. In the Soong *et al.* (1979) morphology, the 10 cm depth would be inclusive of generation 7 airways; as in the Weibel (1963) morphology 16 generations of conducting airways exist within the tracheobronchial (TB) tree. Recent theoretical and experimental studies (Hanna and Scherer, 1982; Nuckols, 1982) support the concept of a graduation in T (to final body temperature) and RH (to saturation) profiles in the upper conducting airways of man for normal breathing in a moderate environment.

As part of a special study, Ingelstedt (1956) cited limited data suggesting that supersaturation might occur under **highly unusual** thermodynamic conditions (breathing in a **cold** chamber) because of "insufficient condensation nuclei" in the inspired laboratory air (0-4°C, 50-60% RH). In a theo-

retical description of "disturbed laminar" flow of particle-free gas through a large airway model (Ferron, 1981) the possibility of transient supersaturation in upper bronchi under more general conditions was envisioned. In an earlier work (Höllander, 1979) a theoretical model of transient supersaturation was also proposed but the model neglected "turbulent" flow in upper human airways; airflow patterns in upper airways are known to be very unstable because of airway branching, the larynx and cartilaginous rings (West and Hugh-Jones, 1959; Dekker, 1961). Ingelstedt and Toremalm (1961) experimentally studied airflow patterns and heat transfer in airway models and concluded that "...the human larynx functions as a constriction in an aerodynamic sense." The larynx, therefore, cannot be omitted from mathematical descriptions of inhaled gases, in part because of its effect on boundary layer development. The boundary layer thickness is of great biological importance because of interchange of heat and moisture between air and mucus membranes. Ingelstedt and Toremalm (1960) postulated the presence of a buffer layer as an interface between a laminar boundary layer and turbulent core flow to simulate upper airway fluid dynamics. Martonen *et al.* (1982) and Martonen (1983a) have theoretically and experimentally demonstrated the influence of laryngeal jet kinetics upon hygroscopic particle behavior.

From scientific principles, Ingelstedt (1956) studying inhaled, relatively particle-free gases postulated "...oversaturation can never occur in experiments at room temperature."; and indeed, supersaturation was never observed in his tests. In a later study, Ingelstedt and Toremalm (1960) again experimentally confirmed that supersaturation could never be recorded during breathing room air. Adapation of Ferron's (1981) model to atmospheres containing particles may be possible if the theory were to include coupling D_C (the diffusion coefficient of water vapor molecules in air) and D_T (the thermal diffusivity of air), and specific treatment of the thermodynamics of gas-particle interactions in the creation of T and RH profiles in human airways. Influences of mixing effects attributable to the laryngeal jet and related flow disturbances *in vivo*, absent in the Ferron (1981) theory, remain as counter-arguments to the plausibility of the phenomenon of supersaturation occurring during the inhalation of ambient aerosols.

Ferron (1977) assumed constant T and RH values for the human bronchial airways to be 37°C and 99.5% for aerosol modeling purposes; the latter RH value can be derived from the osmotic pressure or from the lowering of the freezing point of blood. From theoretical considerations, ambient T and RH conditions inside the human lung have been estimated to be about 37°C and 96%, respectively (Wilson and LaMer, 1948; Milburn *et al.*, 1957). Proctor (1964) has concluded that inhaled air is warmed and humidified sufficiently to reach final body T and 99% RH at the alveolar level; alveolated airways begin at generation 17 in the Soong *et al.* (1979) morphology. Using replica respiratory tract casts, the T of inhaled air at the entrance to the trachea has been demonstrated by Olson *et al.* (1973) to be within 0.2°C of the equilibrium T, 37°C, within the deep lung.

Considering the above factors, two T and RH patterns will be assumed in the current study to provide an envelope of most likely conditions of the influence of hygroscopic growth within the lung upon inhalation of aerosols in a temperate (25°C and 50% RH; Walker *et al.* 1961) environment. To simulate oral breathing, the RH of the trachea will be 90% and will increase monotonically 1% with each airway generation until 99% is attained at generation 9. The value of 99.5% will be maintained for all distal airways. The constant value for peripheral airways is consistent with a state of equilibrium at those levels and is in agreement with the RH levels predicted from theoretical bases, as previously discussed. To correspond to nasal breathing, we assume that RH equilibrium conditions are attained in the trachea and remain at 99.5% in distal bronchial passages. For both modes of breathing we assume the temperature is a constant 37°C in the TB tree, consistent with experimental data quantitating the extent of warming that occurs in the nasal and oral pharyngeal compartments.

Aerosol Deposition. To compute particle deposition probabilities in TB airways, we use a system of equations derived by Martonen (1982) that describes inhaled aerosol behavior in physiologically realistic airflow patterns, laminar and unstable - the latter including the effect of the laryngeal jet.

In a previous study (Martonen, 1983b) the equations have been used to evaluate the suitability of symmetric (Weibel, 1963; Soong *et al.*, 1979) and asymmetric (Horsfield *et al.*, 1971) TB morphologies for representing adult man. Best agreement between theory and experiment was found with the Soong *et al.* (1979) morphology (see Figure 2) which will therefore be assumed here. Depending upon whether oral (90-99.5% RH) or nasal (99.5%) breathing is being simulated, the corresponding changes in size and density of an inhaled ambient particle will be defined by the appropriate curve of Figure 1. Deposition in the TB tree, therefore, will be computed in a manner commensurate with stepwise changes in particle parameters among airways.

RESULTS AND DISCUSSION

Results of nonhygroscopic aerosol deposition computations using the theoretical model agree well with experimental measurements, indicating that *in vivo* particle behavior is being accurately simulated (see Figure 2). Airway diameters were varied in the model to examine the effect of intersubject variability among a population. The theoretical AC (Airway Coefficient) = 1.00 curve signifies total deposition within the Soong *et al.* (1979) morphology. The AC = 0.85 and AC = 1.15 curves are for modified diameters that are decreased and increased in size, respectively. For individual human subjects, the total deposition curves measured by Lippmann *et al.* (1971) had characteristic "S-shaped" curves that within a population had two similar asymptotes and almost parallel, linear segments away from the asymptotes. Significantly, the theoretical model (i) generates the general sigmoidal shapes of such individual TB deposition patterns, and accurately calculates the (ii) slopes of their quasi-linear segments and (iii) positions of their two asymptotes.

The complex effect of assumed RH profiles upon hygroscopic growth and $(NH_4)_2SO_4$ aerosol behavior is demonstrated in Table 1. Two observations can be made from the data: (i) particles in the diffusion-dominated regime have reduced deposition relative to nonhygroscopic particles of identical preinspired size and (ii) hygroscopic growth produces an increase in deposition, relative to nonhygroscopic particles, for particles primarily affected by inertial and gravity forces. For the smallest particles the disparity between nonhygroscopic and hygroscopic cases increases as the magnitude of the average inspiratory flow rate, Q, decreases. This is consistent with the action of diffusion, the efficiency of which is related to the particle residence time in a airway. For the largest particles, however, the opposite occurs because particles grow sufficiently to become effectively deposited by inertial impaction; the efficiency of the mechanism is related to particle velocity. Differences between the two RH profiles are most pronounced at the largest particle size and flow rate tested. In the model an ambient 1.0 μm D_{ae} particle has grown to D_{ae} = 1.4 μm within the trachea for oral breathing (90-99.5% RH), and finally attains D_{ae} = 3.4 μm in generation 10. For nasal breathing (99.5% RH), the 3.4 μm size is assumed in the trachea and all distal bronchial passages. In Figure 3 these findings are explicitly explained in terms of the actions of the dominant deposition mechanisms in the lung for Q = 15 $Lmin^{-1}$.

The effect of an assumed RH profile upon the distribution of deposited H_2SO_4 aerosol mass as a function of inspiratory flow rate is shown in Figure 4. The apparent common feature of (a), (b), and (c) is that increasing the magnitude of the inspiratory flow rate increases deposition in the larger conducting airways while yielding reduced losses in more peripheral airways. Interestingly, the crossover occurs within generations 10-12 for both RH profiles tested.

At the lowest flow rate in Figure 4, total deposition does not vary much between (a), (b), and (c). There is, however, a noticeable shift in mass fraction deposited in larger upstream airways with consideration of the influence of an assumed RH profile upon hygroscopic growth effects. At 60 $Lmin^{-1}$ total deposition increases by a factor of 2 between (c) and (a), and 3 between (c) and (b). Moreover, there is a decided proximal shift in inter-TB distribution at the highest flow rate. In (c), the percent of deposited mass found in upper ($0 \leqslant I \leqslant 5$), middle ($6 \leqslant I \leqslant 10$), and lower ($11 \leqslant I \leqslant 16$) airways is 15.4%, 23.7%, and 60.9%; in (a), the respective values are 20.4%, 37.4%, and 42.2%; in (b), 40.7%, 35.1% and 24.2%.

In Figure 5, RH effects on NH_4HSO_4 aerosol distribution are illustrated. In (a), total deposition is almost the same for a nonhygroscopic aerosol and NH_4HSO_4 assuming oral (90-99.5% RH) breathing. The percent of the total delivered to upper airways, however, has doubled due to growth by water vapor absorption at the expense of dose delivered to the lower airways. With nasal breathing (99.5% RH), NH_4HSO_4 distribution to upper airways is double that for oral breathing; indeed,

distribution is now nearly uniform within the TB tree. The situation is different for the larger particles. Hygroscopic growth in the 90-99.5% RH profile produces a total deposition increase by nearly a factor of 3, but relative dispersion among airways is not so dramatically affected. Growth in the 99.5% RH profile leads to an increase in total aerosol mass deposited by more than a factor of 4 relative to nonhygroscopic aerosols; and is, again, double that for oral breathing.

In summary, the primary factors requiring definition prior to studying possible growth effects are initial particle geometry and density, material hygroscopic growth characteristics, respiratory parameters, and T and RH profiles. The influences of hygroscopic growth on inhaled aerosol behavior are very complex; both the total mass deposited and its regional dispersion may be significantly affected. The factor of hygroscopity, therefore, is an important factor to be included in the hazard evaluation of atmospheric particulate matter of health effects concern.

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TABLE 1. INFLUENCE OF HYGROSCOPIC GROWTH UPON DEPOSITION OF AMBIENT $(NH_4)_2SO_4$ PARTICLES

D_{ae} μm	Q $L min^{-1}$	TOTAL DEPOSITION		
		Nonhygroscopic	90-99.5%RH	99.5%RH
0.3	15	13.4	7.3	7.8
	30	9.9	5.7	6.8
	60	7.5	5.5	7.9
0.6	15	9.4	10.8	13.1
	30	7.2	9.4	13.9
	60	6.4	11.5	20.2
1.0	15	8.8	20.9	26.7
	30	7.5	19.2	30.1
	60	8.4	25.4	44.2

FIGURE LEGENDS

Figure 1. Hygroscopic growth curves of several ambient sulfate aerosols of health effects concern. d and d_o are the droplet diameter and dry particle diameter, respectively.

Figure 2. Comparison of experimental human inhalation exposure data with theoretical calculations.

Figure 3. Relative efficiencies of the inertial impaction (I), sedimentation (S), and diffusion (D) mechanisms as a function of ambient $(NH_4)_2SO_4$ size, D_{ae}. (a) 0.3 μm, (b) 0.6 μm, and (c) 1.0 μm. $Q = 15\ Lmin^{-1}$. Nonhygroscopic particle behavior included for comparison.

Figure 4. The influence of inspiratory flow rate upon the dispersion of ambient 0.6 μm D_{ae} H_2SO_4 particles for different RH profiles in the TB tree. (a) 90-99.5% RH, (b) 99.5% RH. Nonhygroscopic particle behavior included for comparison, (c).

Figure 5. The influence of RH profiles upon the dispersion of NH_4HSO_4 particles. (a) $D_{ae} = 0.4\ \mu m$, (b) $D_{ae} = 0.8\ \mu m$. $Q = 60\ Lmin^{-1}$. Nonhygroscopic particle behavior included for comparison. Airway generation is denoted by "I".

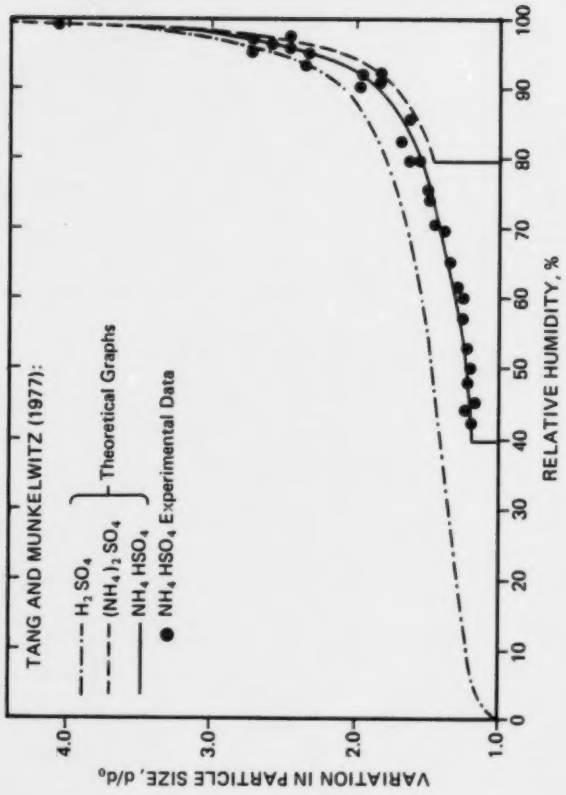
TANG AND MUNKELWITZ (1977):
H_2SO_4
$(NH_4)_2SO_4$
NH_4HSO_4
Theoretical Graphs
NH_4HSO_4 Experimental Data
VARIATION IN PARTICLE SIZE, d/d_0
RELATIVE HUMIDITY, %
1.0
2.0
3.0
4.0
0
10
20
30
40
50
60
70
80
90
100

FIGURE 1.

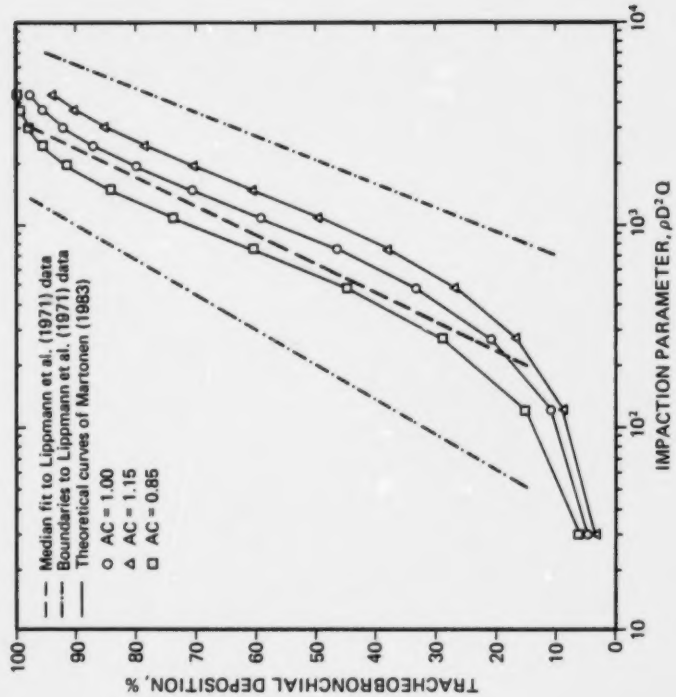
TRACHEOBRONCHIAL DEPOSITION, %
IMPACTION PARAMETER, $\rho D^2 Q$
Median fit to Lippmann et al. (1971) data
Boundaries to Lippmann et al. (1971) data
Theoretical curves of Martonen (1983)
AC = 1.00
AC = 1.15
AC = 0.85

FIGURE 2.

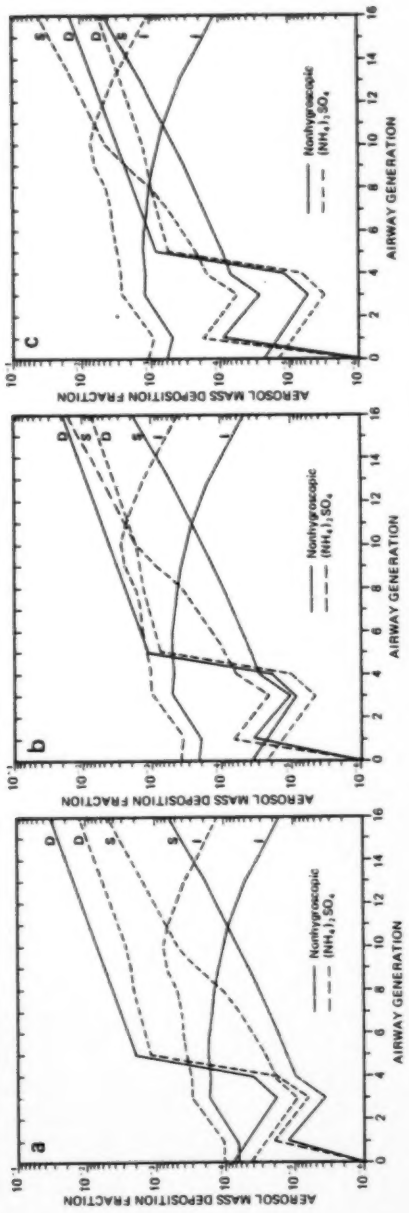
a
b
c
AEROSOL MASS DEPOSITION FRACTION
AIRWAY GENERATION
Nonhygroscopic
$(NH_4)_2SO_4$
D
S
I

FIGURE 3.

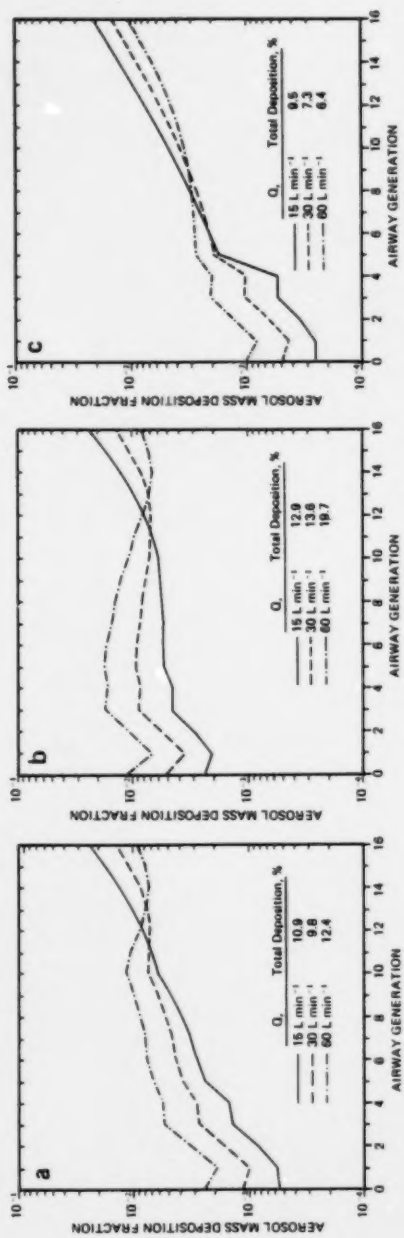
a
b
c
AEROSOL MASS DEPOSITION FRACTION
AIRWAY GENERATION
Q̇ Total Deposition, %
15 L min⁻¹ 10.9
30 L min⁻¹ 9.8
60 L min⁻¹ 12.4
15 L min⁻¹ 12.9
30 L min⁻¹ 13.6
60 L min⁻¹ 19.7
15 L min⁻¹ 9.5
30 L min⁻¹ 7.3
60 L min⁻¹ 6.4

FIGURE 4.

Aerosol	Total %	Relative Dispersion 0≤I≤5	6≤I≤10	11≤I≤16
———	6.6	8.4	20.0	71.6
– – –	7.1	15.0	33.4	51.6
—·—	11.0	34.8	33.4	31.8

Aerosol	Total %	Relative Dispersion 0≤I≤5	6≤I≤10	11≤I≤16
———	7.1	22.1	27.3	50.6
– – –	18.6	19.9	39.0	41.1
—·—	31.6	44.6	35.1	20.3

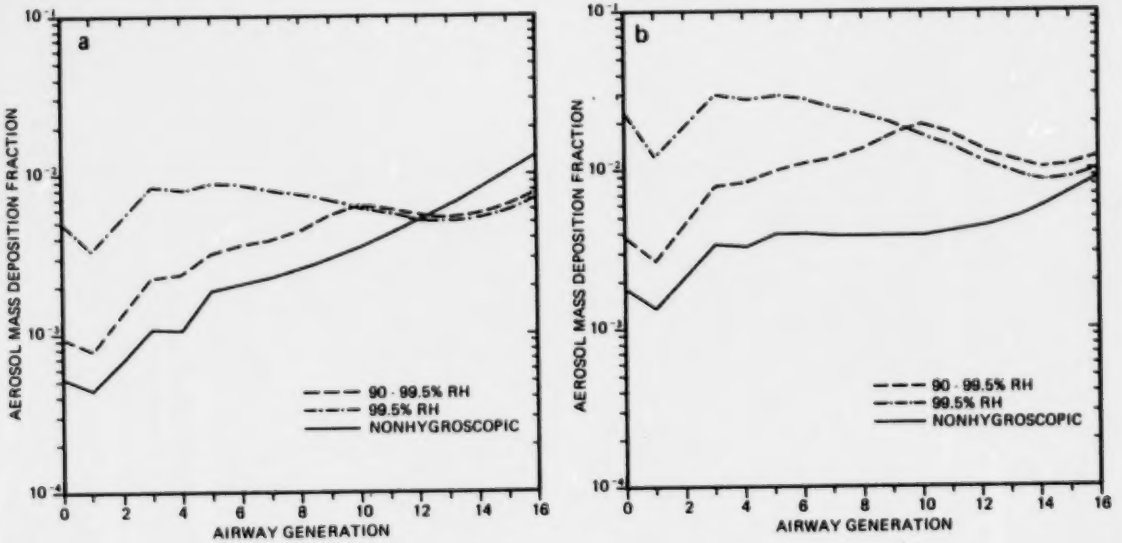

FIGURE 5.